Special Article

NOTES ON ENCEPHALITIS LETHARGICA IN SAN FRANCISCO.

By PHILIP KING BROWN, San Francisco.

Encephalitis Lethargica has made its appearance on the Pacific Coast in numbers sufficient to emphasize the epidemic character of the disease, and in a guise that frequently detracts attention from the chief characteristics of the disease as thus far described.

Ophthalmoplegia, blurred vision, listlessness or stupor, a droop of one or both lids, distorted facial muscles, spastic, tremulous, slow movements, difficult urination, increased or absent knee and patella reflexes, fever, absence of leucocytosis, and slight increase in spinal fluid cell count (mononuclear forms), have marked most of the cases. The stuporous state may be so deep that patients cannot be aroused. The death rate is forty per cent. Pain has not been reported as a conspicuous symptom, and is referred to as wanting by nearly all writers.

The cases that have passed under my observation since August 24, when the first one was brought to San Francisco from Reno, Nevada, have presented a range of symptoms marked by a variation, from an *extremely prolonged cycle covering from four to six months,* to several where death has ensued in four to eight days. The acute cases have presented all characteristic symptoms of the classical disease *to which has been added in the majority shooting pains of agonizing nature accompanied by muscular spasm.* The pain is not always in the part involved in the spasm, although most commonly so, and the pain may cease long before the spasm.

The following brief abstracts are given, by no means representing a complete study of the cases, but serving to show the protean nature of the symptoms.

H., J. E. Age 46. Locomotive engineer. Entered hospital August 25, 1919. Illness began with headache, nausea, general malaise, weakness on left side of body, some incontinence of urine, marked constipation, stiffness of back. Some pain in left hypochondriac region (localized). Patient states (two weeks later) that he thinks it all due to exposure and heat while at work. Patient in stuporous condition, aroused only with difficulty. Answers questions slowly and in a dazed way. Pupils pin point, equal, react to light and accommodation. Neck: very little tenderness and stiffness; some rigidity and stiffness of back. Kernig's sign: negative. Reflexes: K. K. plantar, triceps, biceps and periosteo-radial, active. Delirious at night. Threatened to kill night nurse. Wife reports patient has been doing irrational things for one year. Urinates on floor at night. Cannot be made to understand what to do. Moody and depressed. **For last year has slept inordinately, i. e., all day after sleeping well at night.** Wassermann: negative on blood and spinal fluid. No evidence of brain inflammation, no abnormality in C. S. fluid except 32 cell count and plus pressure. No nystagmus. For a few days complained of dizziness chiefly when head is turned up and back to right, although **any sudden movement of eyes brings it on.** No nausea. Complains that left leg is weaker than right. More spastic in right than left. Knee reflexes very lively but equal. Plantar responses not normal in either foot, but no typical Babinski, and no Gordon, Oppenheim, etc.

September 11. States he was never entirely unconscious. Before the "cloud" settled on him he noted a general weakness, and especially in left leg. Left arm and hand weaker than right. Voice still monotonous. Distinct but slight internal strabismus. States that for two months he has seen double at times.

October 24. Patient has improved steadily. Spasticity is less and vertigo has nearly disappeared. Clear in mind and has shown no unsocial condition today. Acute disorder of July and August over.

J., M. L. Age 57. Male. Examined with Dr. Coffey, October 17, 1919. States he passed three nights without sleep, following day was much exhausted. Slept heavily that night. In a. m. found he saw double, no headache, perfectly rational; weak, but able to walk. **Examination:** Temperature 102.8, Pulse 96, Resp. 18. Markedly staggering gait with weakness on right side; marked tremor suggesting cerebellar disturbance. Slight motor weakness of muscles of right face. Coarse tremor of part brought into play in attempting any muscular movement, as frowning, closing eyes, protruding tongue or even showing teeth. Grip not strong in either hand. Hands and feet, particularly right, move constantly. Pupils dilated slightly, do not react to light. Cerebration slow. Tremor and attitude very like man befuddled slightly by alcohol. **Eye Examination:** Pupils small and probably do not react to light. No nystagmus. Slight internal strabismus right eye. No other involvement of cranial nerves. He sees better, but still sees double. Blood and spinal fluid negative for syphilis. Widal: negative. Cutaneous sensitiveness diminished over chest but not elsewhere. Both ankle jerks and right knee jerk absent. No Babinski. No disturbance of sphincters. Cerebration distinctly slow, but he understands and executes movements after they are repeated to him several times. A marked conjunctivitis from start. Wife reports he is most of time in a drowsy state, but can be aroused with no great difficulty. With exception of headache there has been no pain associated with attack.

November 6. Wife reports patient delirious at times, although afebrile for several days. Able to walk with less vertigo. Blurred vision in left eye still present.

Mrs. G. F. B. Age 36. Clerk in bank, widow with two children. Seen October 17 with Dr. Wymore. On October 11 felt slight steady pain in right side of neck. Next day it was more stabbing and constant. October 14 and 15 pain in neck increased. On October 16 went to work, but pain became paroxysmal and extended down arms and to breast, and back. Paroxysms were accompanied by spasm of muscles in region involved. Entered hospital October 16 in violent paroxysmal pain which continued uninfluenced by drugs for four days. Then for three days had no pain except in ball of left foot, but there continued in abdomen violent twitchings due to contraction of diaphragm. This is quite rhythmical and largely involves the right side, but occasionally the left. What caused relief of pain is not clear; morphia and pantopen did no good and made her even more excited. Nothing brought relief for first two days; antipyrene, salipyrene, aspirine alone and with phenacetine, caffeine, bromide and chloral, veronal, etc., all did no good. The first relief was

from 1/150 scopolamine, and for two nights this helped. Luminol was tried in dose of 1½ grains every six hours, and dialciba two tablets at night. This gave fair nights, but no relief of spasm. There was slight fever. On one occasion there was a slight amount of reducing substance in urine. Patient quite depressed. No previous eye disturbance, but sees double. No disturbance of hearing. No knee or ankle jerks. For several days past noticed considerable difficulty in getting flow of urine started, as though a spasm might be acting there. She has menstruated excessively recently, had influenza just a year ago. Has been said to drop things from both hands more easily lately. October 17 patient complained of a constant desire to urinate and move her bowels, and had great difficulty doing either one. Depressed all day. Pulse good. Unable to eat and nauseated by solid food. Very clear in her mind. States she sees double; there is slight strabismus. Tongue protrudes straight. No other involvement of cranial nerves except that smell and taste have been lost since influenza. Patient died quite suddenly at 1 a. m., evidently from paralysis of respiration.

C., T. Age 60. October 16, 1919. Began to see double after three or four days of insomnia, since then is sleepy all the time, irrational when aroused. Complained of pain in back of head and neck. Spells when he becomes rigid. **Examination:** Drowsy and irrational. Reflexes equal and lively in arms and legs. Pupils react to light and are equal. Does not raise left eyelid. Very resistive to passive movement. Wife states eyes were crossed before entrance to hospital. Conjunctiva injected. Rectal tempt. 101. Blood pressure 160/90. Wh. bl. count 14,100, 82% polys. Had to be tube fed the last five days and no sphincter control for last six days before death. **Course in hospital**: Ptosis of left lid continued until death. Progressive stupor. Paresis muscles of left face. Spinal fluid contaminated with blood. Wassermann negative. Died on ninth day.

C. E. D., Mrs. Age 31. Seen with Dr. Burnham. October 23, 1919. Neuritis for 18 days; began in back of neck and extended to both shoulders and down arms to hands. Pain spasmodic, agonizing in character and accompanied by muscular spasm where it is felt. Has felt it slightly for one week in both eyes, and has noted a progressive diminution in vision for five days. Temperature 100-100.5. No apparent lethargy. No obvious initial infection. Influenza last October; husband states she has not been well since, markedly unwilling to exert herself, a great contrast to her usual self. Pain on right side of throat has caused some difficulty in swallowing for a few days. **Examination:** Ptosis of left eye, spasm on left side of face at intervals of from 3 to 10 seconds. Synchronous with this is an intense pain in right eyeball, right side of nose and right breast region. Pupils equal and react to light. No knee or ankle jerks. No Babinski. Tache cerebrale marked. Restlessness of lower limbs, particularly the left, which in addition is flexed each time there is a spasm in the left pectoral region, but no pain is felt in legs. Spine not rigid, neck not stiff. No Kernig sign. Bilateral nystagmus, stronger to the right. Tongue deviates to the left. Slight weakness to lower facial group. No tremor or incoordination. Herpes labialis. Hallucination of sight, and it is obvious that only the intense pain keeps her from a drowsy state. The pain spread down the arm, and four days later was occurring in waves in two fingers of left hand, but without spasm. Spasms and pains in breasts were less severe and less frequent. Arm reflexes gone but right knee reflex returning. A late development was a rapid heart and brief attacks of syncope.

November 11. Afebrile for seven days; somnolent but free from pain. Is on digitalis for heart condition.

D. C. American. Age 19. Single. Seen with Dr. J. J. O'Connor. Patient somewhat irrational and slow of speech. On October 10 after a dance, complained of neuralgia over left side of face and forehead; on October 11 began to vomit after taking any solid food. Quit work October 14 on account general weakness. October 15 began to have hallucinations, sings and makes speeches. Several days ago had neuralgia in left arm and left side of scrotum. Is a trifle slow in his actions and responses. Says he sees double. **Examination:** Drowsy condition, answering questions very slowly. Facial expressions normal, no paralysis. Pupils unequal, left smaller; react to light and accommodation. Slight **horizontal nystagmus to right.** November 5. Nurse reports respirations slower when asleep, but accelerated by talking. During examination they number 36, and suggest a labored effort like a person after exertion. Nothing in lungs to account for this. Upper extremity reflexes equal, also exaggerated abdominal. Suggestion of weakness in left extremal rectus and left face muscle. Is a trifle slow in his actions and responses. Knee jerks very lively. Ankle clonus both sides. **Oculist's report:** Some blurring of outline of nerve heads. Suggests toxic condition rather than a change due to intracranial pressure. October 19. Wh. cell count 14,100. November 4. Wh. cell count 12,400. Blood serum and cereb. spinal fluid give Wassermann test negative. November 11. The course of the disease is sub-acute. Fever of 1-2 degrees for first ten days in hospital. None since. Hallucinations were brief. Present state marked chiefly by somnolence.

S., F. Italian. Age 47. Single. Seen with Dr. L. D. Bacigalupi. September 24, 1919. Patient began having pain in arms, hands and back, and two days later in elbows, wrists and finger joints. Finger joints swollen and very painful; the swelling has been coming and going since. Pain constant since beginning. No pain in knees or ankles. Pain in hips. Says he sweats all the time. No headaches. Complains of pain in arms and back. Daily fever to 101 degrees. Pupils react well to light and accommodation. Slight nystagmus. **Neuro-muscular**—No atrophy. Tendon reflexes are exaggerated. No clonus or Babinski. Patient perspiring slightly. October 10. Patient has been in a stuporous condition several days, and has developed a paralysis of left side of face. Cannot wrinkle left forehead or close left eye. Whole 7th seems involved; 5, 6, 8 and 9, 10, 11 and 12 O. K. October 25. Great restlessness for at least two weeks, wanders about at night when not restrained. Lies in comatose condition the rest of the time, beginning about the 12th and steadily deepening. Speaks when aroused, but confused from the first. Marked conjunctivitis. Slight palsy left lid. Tongue protrudes slightly to right. Palsy left face muscles. Reflexes of extremities equal and normal. Blood serum and cerebro fluid are Wassermann negative. November 11. Patient afebrile for two weeks; recovering slowly. Lies in semi-somnolent state all day, but is more easily aroused.

K. J. Age 23. Attorney. Seen with Dr. C. H. Thompson. October 29, 1919. **Complaint:** Pains in back of neck; intense neuralgic pains down both arms in paroxysms accompanied by spasms of muscles beginning ten days ago. Within three days vision slightly blurred and saw double. Temperature 101-105. Pulse about 100. Respiration 25-30. No trouble with bladder or rectum. Lies in semi-stupor, but easily aroused. No nystagmus. Slight strabismus. Reflexes in upper extremities exaggerated but equal. Knee jerk in left leg but not in right. Pain and spasm have decreased in frequency and severity for three days. Temperature still 100-102. No leucocytosis. Spinal fluid cell count 20. Wassermann negative. Urine normal. November 3. Still lies in stupor, but more easily aroused. Distinct tendency to ankle clonus. Plantar reflexes normal. Afebrile for two days.

G. G. Age 44. Telegrapher. November 2, 1919. After exertion in running for train noted tingling in left face and above and behind left ear. In few minutes throbbing, sharp twitching began in same region; following day extended to left shoulder and within 24 hours slowly extended to left arm, forearm and hand. The shooting pain was rhythmic and accompanied by contraction of muscles in part affected. Vision blurred and saw double on third day. No bladder, rectal, digestive or locomotor symptoms. Wife noted he was cross-eyed early in the trouble. Began to be drowsy about sixth day and remained so six days; had rambling delirium for few days. **Examination:** Double nystagmus, slight droop to left lid and left extremities. Upper extremity reflexes exaggerated except left triceps which is wanting. Lower extremity reflexes sluggish but equal.

Medical Building.

Original Articles

INTESTINAL FLAGELLATES: A PLEA FOR THEIR PATHOGENICITY.

By JOHN V. BARROW, S. B., M. D., Los Angeles.

A general survey of the literature on flagellated protozoa impresses one with their growing importance. Twenty years ago, they were reported in different human organs as a sort of curiosity, but scarcely to be thought of as pathogenic parasites. However, very recent years have brought a greater realization of their importance as producers of pathology. The amoeba and ciliated balantidium coli were first to gain recognition. Now they have a young army of medical men ready to respect their pathogenic power. What has happened in the role of the amoeba, and balantidium, is beginning to be realized in the case of the flagellated monads.

The influence of the "harmless commensal" literature is much to be regretted as it has doubtless deprived the profession of the report of many cases of pathology due to these parasites.

The conviction of most recent authors has been based almost exclusively on the ability of these organisms to produce dysentery. This is well illustrated by the work of Escomel (1) who from Peru cites one hundred and fifty-two cases due to trichomonas intestinalis. This infection was borne by polluted water. Mello-Leitao (2), Derrieu and Reynaud (3), and Rhamy and Metts (4), all report cases in which dysentery is given as the characteristic pathology. The latter authors even go so far as to state that they never have seen the flagellates in the intestines without their producing dysentery. Their review of a dysentery epidemic due to flagellates is very instructive. There were seventy-eight cases in all with eighteen deaths. The epidemic was traced to impure water. The clinical picture was diarrhea with colicky pains, watery or slimy blood-stained stools, weakness, dyspnea, loss of weight, anemia simulating the pernicious type, jaundice, and urticaria. Later the stools consisted of blood, pus, and active trichomonads. He noted a moderate eosinophilia. Ulcer of the rectum occurred once.

Prentiss (5) from El Paso, Texas, reports several cases of diarrhea both chronic and acute, in which the cercomonas hominis was the causative factor. In his two autopsied cases there was intestinal catarrh but no ulceration. Chatterjee (6) considers flagellate dysentery a certainty and cites seventy cases in India as proof.

Nearly all the tropical writers emphasize dysentery as the only clinical manifestation of the presence of flagellate infection. Houghwout (7) refreshes us with a broader conception in his most excellent critical review from which I have drawn largely. He cites a case from St. Luke's Hospital, Manila, in which he considers the trichomonas was responsible for the diarrhea, mucous stools, and a small rectal ulcer. He offers no proof that the flagellates were the tissue invaders. His conclusions suggest so well a reason for the clinical picture which so often accompanies "Flagellosis," that I think they should be emphasized here. The effects that might be looked for, are summed up under these six heads:—

First: The production of antigrowth vitamines or growth inhibiting substances.

Second: The production of substances directly toxic.

Third: The unfavorable effects upon the host through the liberation of the metabolic products of the parasites.

Fourth: Mechanical irritation of mucous surfaces.

Fifth: Interference with absorption in the intestine through adherence of large numbers of the parasites, as is the case with lamblia infection.

Sixth: Actual invasion and destruction of tissues with concomitant sequelae.

The above author makes no attempt to prove these points, but clinical findings from my own series of cases are plentiful to substantiate every statement.

The above six points may well be grouped into two or three larger headings, such as, metabolic, toxic, and mechanical disturbances.

With some of my own cases, I wish to show the applicability of these principles. The following table will be more illustrative than a laborious description of a series of twenty-one cases.

In the table, it will be noted that there were three cases of intestinal perforation. From one of these the trichomonas intestinalis was recovered, both in the feces and in the peritoneal drainage. Amebic cysts were present in small numbers in the stool. Involuntary diarrhea lasted while peritonitis was present. The organisms were in overwhelming numbers. The scarcity of the amebae found as against the abundance of monads, could not but impress the observer that the latter and not the former were the real offenders. The other perforating cases were heavily infected with monads while no active amebae were seen. The entire series demonstrates focal bowel tenderness in such a way as to stimulate the belief that one is dealing with early possible perforating spots. The main points of tenderness are colonic and most